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Quantifying the effect of age and contraction mode on the force-velocity-power relationship in the knee extensors

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Graduate Program in Kinesiology

A thesis submitted in partial fulfillment of the requirements for the degree in Master of Science

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**QUANTIFYING THE EFFECT OF AGE AND CONTRACTION
MODE ON THE FORCE-VELOCITY-POWER RELATIONSHIP IN
THE KNEE EXTENSORS**

(Thesis Format: Monograph)

By

Justin Roland Paturel

Graduate Program in Kinesiology

A thesis submitted in partial fulfillment
of the requirements for the degree of
Master of Science

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The University of Western Ontario
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ABSTRACT

PURPOSE: To assess and compare the force-velocity-power relationship (F-V-P) using isotonic and isokinetic knee extensions in 11 younger (19-30y) and 11 older (69-81y) men. **METHODS:** The construction of the F-V-P curves were attained with each participant performing maximal, explosive knee extensions at 8 different loads (isotonic) and 8 different velocities (isokinetic) on a Cybex HUMAC NORM dynamometer. Maximal voluntary contraction (MVC or P_o), maximal shortening velocity (V_{max}), maximum power, optimal torque (P_{opt}) and velocity (V_{opt}) were assessed and compared. **RESULTS:** The older men were 43% weaker, 18% slower and ~54% less powerful than the younger men for both isotonic and isokinetic contractions. For the older men, P_{opt} was 35% and 38% lower in the isotonic and isokinetic F-V-P relationships, respectively. The V_{opt} of the older men were 36% and 25% slower, respectively in the isotonic and isokinetic power curves. Interestingly, the V_{opt} achieved during isokinetic testing was 16% and 36% higher for both younger (400 vs 346°/s) and older (300 vs 221°/s) men, respectively, compared to the isotonic V_{opt} . **CONCLUSION:** For the isotonic modality, the weaker and slower knee extensors of the older men contributed equally to lower power whereas for the isokinetic modality, age-related muscle weakness contributed more than velocity to the reduction in power. The muscle behaves differently between modalities, which may explain parts of the results. The isotonic modality better modelled the F-V relationship, with fewer inherent limitations compared to the isokinetic modality.

Keywords: Isokinetic, Isotonic, Dynamic, Aging, Quadriceps Femoris, Torque-Angular Velocity, Maximum Power

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~In life, if you get all tangled up just keep tangoing on~

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LIST OF ABBREVIATIONS

F-V-P – Force-velocity-power relationship
MVC (P_o) – Maximal voluntary isometric contraction
P – Force
 P_{opt} – The optimal force
 P_{opt} / P_o – The relative optimal force or load compared to the MVC (P_o)
V – Velocity
 V_o – Unloaded shortening velocity
 V_{max} – Predicted maximal shortening velocity
 V_{opt} – The optimal velocity
 V_{opt} / V_{max} – The relative optimal velocity compared to V_{max}
 P_t – Peak twitch amplitude
TPT – Time to peak twitch torque
 T_s – Superimposed twitch
 T_r – The resting twitch
HRT – Half relaxation time
RTD – Rate of torque development
VA – Voluntary activation
MHC – Myosin heavy chain isoform
ATP – Adenosine triphosphate
ADP – Adenosine diphosphate
Pi – Phosphate ion
TNF- α – Tumor necrosis factor- α
IL – Interleukins
IGF-1 – Insulin-like growth factor -1
ROM – Range of motion
ANOVA – Analysis of variance
SD – Standard deviation
E-C coupling – Excitation contraction coupling
N/A – Not applicable
ITT – Interpolated twitch technique
EMG – Electromyography
SEC – Series elastic component

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CHAPTER 1

1.0 Literature Review

1.1 General Introduction

Through the intimate connection between the nervous and musculoskeletal systems animals can do physical work in the external environment. Much of the foundation towards our understanding of muscle physiology was developed throughout the twentieth century: force-velocity (F-V) relationship (1930s), sliding filament theory (1954), cross-bridge mechanism (1957), length-tension relationship (1960s), fiber typing (1960s), motoneurone size principle (1965), muscle fatigue (1960-1980s), and the concept of sarcopenia (1988). Many of these earlier discoveries were performed in animal models, whether *in vivo*, *in situ*, or *in vitro*, and the applicability or direct relationship to human systems physiology and function is not well-known or has limitations for experimental testing. Nevertheless, evaluating the findings from reduced preparations (both animal and human) to the *in vivo* human organism enables corroboration of previous findings or new insight into how joint biomechanics, muscle architecture or voluntary muscle activation influence muscle performance.

The F-V relationship is a central tenet in understanding muscle function as it dictates muscle performance, setting the limits of maximum force, speed, and power. A comprehensive understanding of the many factors influencing this relationship will improve training, rehabilitative, and clinical modalities in exercise, athletics, health, aging and disease. Many of the studies on aged-related alterations to muscle function only assessed maximal strength or limited dynamic conditions inadequate to assess the F-V relationship [1-15], whilst of those investigating the F-V relationship in aged humans,

few have applied Hill's equation [16-19] and others reported linear-like relationships or didn't report muscular power results [20-27]. Recent studies have emphasized the importance of muscle power, the product of force and velocity, for test outcomes and quality of life among the older population [14,28]. A concomitant reduction in strength and speed will exacerbate age-related power loss compared to either factor alone, with particular emphasis on speed decrements having a greater impact on functional outcomes [29,30]. Investigations of the functional consequences of sarcopenia are plentiful among animal [31-36] and human [1,2,37-42] models, but the interrelationships of various contributing factors to sarcopenia are not fully understood.

There have been various methods and equipment used for testing strength, speed, and power and therefore it is difficult to compare outcomes among studies. Among the initiative, customized equipment for the elbow flexors [43] and knee extensors [44] have been devised, as well as the Nottingham power rig, assessing the leg press movement [26]. Since the 1960s and 1970s commercially available equipment such as multi-joint dynamometers (i.e., Biodex & Cybex) have been utilized to assess human muscle strength, speed, and power [22,45-50]. Early dynamometer models offered an isometric and isokinetic contraction mode, thus, many of the dynamic studies were limited to the isokinetic mode to assess muscle power. However, recent commercially available models offer an isotonic mode, which better simulates normal dynamic contractions because the load or resistance is often constant and the velocity of joint rotation can vary. Despite some earlier studies that used isokinetic dynamometers, there has been renewed interest to move beyond isometric contractions, in the field of muscle function and adult aging, to dynamic contractions (both isokinetic & isotonic), directed to some degree at

understanding the influence of shortening velocity on power production [27,51]. An understanding of basic dynamic contractile function with aging will provide a solid basis for testing and exploring the effects of fatigue, training or rehabilitation on an aged population.

1.2 Quadriceps Femoris: Structure and Function

1.2.1 Gross Anatomy, Architecture and Composition

The quadriceps femoris (QF) is a group of 4 separate large muscles all with a common insertion onto the patella and through the patellar ligament attach to the tibia. The vasti muscles (vastus medialis, vastus lateralis (VL), and vastus intermedius), aptly named for their size and location, originate from various aspects on the femur, only crossing the knee joint, contributing to knee extension. On the other hand, the rectus femoris originates on the pelvis (anterior inferior iliac spine), thus crossing the hip and knee joints, contributing to both hip flexion and knee extension [52].

The pennation angle for the VL have been approximated between 14 - 20.3° [53-56]. The reported fascicle lengths range from 65 – 129mm [53-55,57]. The cross-sectional area for VL and QF range from 18 - 36 cm² and 65 - 88 cm², respectively [53,56,58,59]. Apportioning fiber type based on myofibrillar ATPase (mATPase), fiber types, mainly for the VL have been classified as: Type I: ~45%, Type IIA: ~ 35%, and Type IIB: ~20% [60-62]. Myosin heavy chain (MHC) isoforms have a strong correlation with mATPase with distributions as: MHC-I: 37%, MHC-IIA: 41%, and MHC-IIB: 22% [62].

1.2.2 Isometric Neuromuscular Properties

The estimated optimal knee angle for isometric force production from an *in vivo* length-tension relationship for the QF is $\sim 90\text{-}100^\circ$ (knee fully extended = 180°) [21,25,63]. The average strength of the QF among the young men and women approximate $\sim 270\text{N}\cdot\text{m}$ and $175\text{N}\cdot\text{m}$, respectively, during near maximal voluntary activation, $\sim 95\%$ [22,45,48,64,65]. Also, the motor unit firing rates generated during voluntary efforts are similar for both VL and VM in young men, ranging from 8Hz at very low efforts and increasing to 26Hz for maximum efforts [9,66].

1.3 Muscle Physiology

1.3.1 Force-Velocity-Power Relationship

Initial studies by A.V. Hill [67], using an isolated sartorius muscle from a frog, investigated the energy (heat and work) liberated during muscle contractions, isometrically and dynamically. His observations led to the description of the force-velocity (F-V) relationship as a rectangular hyperbola with the following formula:

$$(P+a) \cdot (V+b) = (P_o+a) b \quad (1)$$

Force generated during a maximally activated static contraction (i.e., zero velocity/shortening) is P_o . As the muscle is allowed to shorten with faster velocities (V), the force generated (P) declines. The force (a) and velocity (b) constants can be calculated through linearizing equation (1), and fitting a linear least-square regression line (refer to appendix A). The ratio between (a) and P_o (a/P_o) reflects the concavity of

the curve; a greater ratio reflects less curvature, which equates to a more powerful muscle [67]. The intercept of the velocity axis, coinciding with zero load imposed onto the contracting muscle is maximal velocity (V_{\max}). The construction of a F-V curve requires a series of measurements of muscle contractions. Initially, the P_o is measured by a maximal tetanic isometric contraction. Next, controlling the load (constant force-variable velocity; isotonic) or velocity (constant velocity-variable force; isokinetic) and measuring the other contributing variable, (i.e., velocity or force, respectively) during a dynamic contraction provides a data point for the curve. Plotting these data points across many loads (isotonic) or velocities (isokinetic) will create a relationship, see Figure 1. Since the pioneering work of A.V. Hill there have been many investigations of the force-velocity relationship within animal [68-76] and human [17,19,23-25,43,44,48,50,77,78] models.

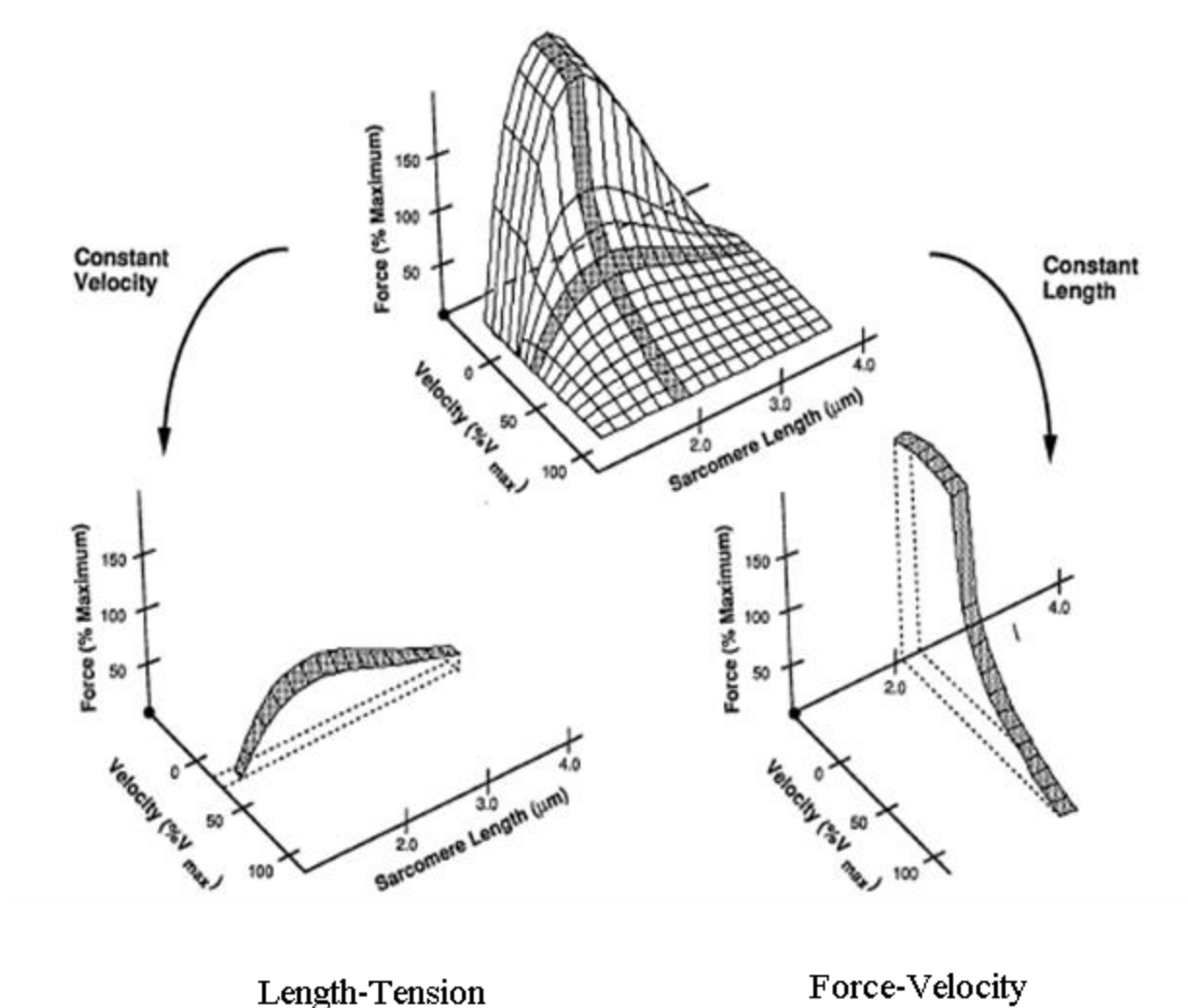


Figure 1. An integrated model of the length-force-velocity relationship. *Adapted from Friden et al. Med Sci Sports Exerc 24: 521-30, 1992*

In vitro and *in situ* preparations best represent the hyperbolic F-V relationship described by Hill [67], as utilizing whole muscle or individual muscle fibers allows more control of the experimental conditions and eliminates many confounding, albeit important, factors compared to *in vivo* human experiments. Reduced preparations behave as expected with the plotted data tightly coupled to the modelled F-V curve from Hill's equation [69,74,79,80]. Experiments of human *in vivo* F-V relationships present many challenges. Muscle architecture [17,50,81-83], joint biomechanics [84-87], heterogeneous muscles [47,74], and central nervous system input [88,89] all influence the performance of muscle. These factors likely cause the experimental F-V relationship to deviate from the classical rectangular hyperbola F-V model as many studies report linear-like F-V relationships. Among the earliest to investigate the *in vivo* F-V relationship of human muscle, Wilkie [43] assembled a custom built isotonic apparatus for the elbow flexors, demonstrating the hyperbolic F-V curves modelled well with Hill's equation. The earliest to use the isokinetic dynamometer to measure force, velocity, and power in human knee extensors were studies by Thorstensson et al. [90], Perrine & Edgerton [48], Froese & Houston [91], and Prietto & Caiozzo [49] all of which reported F-V relationships not resembling the classic hyperbolic curve. Recently, of the reported isokinetic F-V relationship studies, two of the knee extensors reported linearly shaped F-V relationship [22,25], while others reported the hyperbolic curve expected from Hill's equations for the adductor pollicis [77,92,93] and plantar flexors [17,94]. Apart from Wilkie [43], few studies have utilized an isotonic modality to investigate the F-V relationship in humans. Similarly to the inconsistencies observed with the isokinetic modality, some reported a hyperbolic isotonic F-V curve for the elbow flexors [18,19,95]

and knee extensors [44], while others did not for the knee extensors [24], plantar flexors [20], and dorsiflexors [27]. Differences in methodology among these studies likely explain the lack of consistency and the rather poor approximation to the classic F-V curve. A potential limitation to human *in vivo* studies is the capacity of full activation of the contracting muscle within the prescribed range of motion. Investigating the F-V relationship of the knee extensors via voluntary unrestrained, voluntary isometric-release, and femoral nerve stimulation contractions, James et al. [96] demonstrated electrical stimulation elicited reliable curves, however could not be fitted with Hill's equation, agreeing with other studies [97,98]. However, the torque recordings from James et al. [96] were angle-specific (the most widely used torque value for isokinetic modalities) which has been scrutinized as being an inappropriate method to assess torque [99,100]. Angle-specific and peak torque have been the most common reported torque values isokinetically, however, Rácz et al. [99] proposed measuring the mean torque throughout an isokinetic contraction as the input value for the F-V relationship. Furthermore, the characteristics of the isokinetic contraction were modified to behave more so as an isotonic contraction, that is, the velocity linearly increased to the preset velocity rather than a constant velocity [99]. In conclusion, there are two primary contributors to the equivocal reports of the *in vivo* human F-V relationship, apart from the modality selected (i.e., isotonic vs. isokinetic): different laboratory equipment used for testing, and differing data analysis methods implemented for calculating the F-V relationship.

1.3.2 Determinants of Shortening Velocity

The most useful predicted property of Hill's equation is that related to the maximal shortening velocity (V_{\max}). In 1979, Edman developed a technique (the slack test) to directly measure shortening velocity at zero load, termed unloaded shortening velocity (V_o). While an anchored muscle fiber is maximally activated isometrically during a tetanic stimulation, a quick release of the lever arm allows the muscle fiber to shorten ahead of the sliding myofilaments, causing tension to drop to zero. Plotting the time required for force redevelopment against various magnitudes of quick release provides a quantitative estimate of V_o [80]. V_{\max} is consistently lower than V_o , because it is a prediction reflective of a composite of all fiber types within a muscle, as opposed to V_o , which selectively measures the MHC-II fibers [71]. Shortening velocity is one of the most distinguishable features among the various types of muscle fibers, with the order of fastest to slowest: IIX > IIA > I, and up to a 4-fold difference [79,101].

From Huxley's [102] cross-bridge theory, the shortening velocity is dictated by two components: the length of the power stroke and the duration of attachment. The duration of cross-bridge attachment is influenced by the rate constants of the enzymatic reactions between different steps in the cross-bridge cycle [103]. Recently, the release of ADP has been established as the limiting step to shortening velocity, particularly among the MHC-II group [104]. If shortening velocity is dependent on the cycling of cross-bridges and not total amount of cross-bridges, then studies lowering available cross-bridges via change overlap of myofilaments or reducing calcium concentration will have little effect on shortening velocity. Edman [80] altered the length of the muscle fibers and available calcium to parameters that drastically reduced isometric force, however shortening

velocity was unaffected. Barany [105] demonstrated that shortening velocity was positively correlated with myosin ATPase activity, which was shown later to be related with MHC isoforms [106]. The shortening velocity of the muscle fibers MHC isoforms have a positive correlation as well as similar sensitivity to increases in temperature [107,108].

In vivo whole muscle architecture affects shortening velocity as well. Muscle fibers that are not aligned with the direction of pull, such as pennated muscle, shorten in a two-axis plane rather than parallel to the muscle. The behavior of muscle fibers take advantage of this architectural set-up by establishing a ‘gearing’ factor, that is, the shortening velocity at each hierarchical level of the whole musculo-tendinous unit differs to optimize performance [83]. Also, Lieber and Ward [86] illuminated the importance of muscle fiber lengths and joint moment arm to the *in vivo* muscle performance. Longer muscle fibers (greater number of sarcomeres in series) and smaller joint moment arms typically have faster shortening velocities than shorter muscle fibers or larger joint moment arms [50,86].

1.3.3 Determinants of Force Production

Unlike shortening velocity, force is wholly dependent upon the number of strongly-bound cross-bridges engaged. Thus, strategies to engage more cross-bridges will result in more force. Gordon et al. [109] established the length-tension relationship, demonstrating that the tension is directly related to the amount of overlap between the myofilaments; with tension peaking $\sim 2.2\mu\text{m}$ and decreasing at shorter and longer lengths [109]. With

regards to cross-bridge kinetics, it is advantageous to have a lower detachment rate constant and a higher attachment rate constant. The isomerization of a weakly-bound cross-bridge AM-ADP-Pi with sequentially releasing Pi will create a strongly-bound cross-bridge which increases the affinity of more cross-bridge formation via influence on the troponin-tropomyosin complex [110]. Specific tension (unit of force/unit of area) of muscle fibers appears to be modestly dependent modestly on MHC isoform. Although not as pronounced as V_{\max} or peak power, typically MHC-II fibers have a higher specific tension (up to 50%) than MHC-I [79,111], while others report no difference between MHC-isoforms [1,112]. Calcium kinetics are a major contributor to force production, mainly to expose the binding site of actin for the cross-bridge. Higher calcium concentrations in the sarcoplasm translate into a higher force, twitch or tetanic, as dictated by the force- Ca^{2+} relationship [113]. Furthermore, as the mechanism of calcium release from the sarcoplasmic reticulum is governed by the rate of action potentials from the motoneurons, increasing the motor unit discharge rate to the muscle will achieve higher calcium concentrations (temporal summation), whereas recruiting more motor units engages more cross-bridges (spatial summation).

With regard to whole muscle, larger muscles generate more force than smaller muscles. Thus, heavy resistance training to increase muscle mass (cross-sectional area) translates into more contractile protein (sarcomeres in parallel). Additionally, the shape of the muscle will influence the force generating potential (i.e., fusiform/parallel vs pennate). Pennate muscles allow more contractile mass per cross-sectional area thus increasing the physiological cross-sectional area. However, continued muscle hypertrophy causes greater pennation angle (creating a greater vector angle) and thereby reducing the force

translated to the joint [114]. The biomechanics of the joint will greatly modify the torque production. For example, an insertion further away from the fulcrum of the joint will increase the moment arm, and subsequently torque potential. Thus, the interrelationship between the muscle architecture and joint biomechanics defines its function during muscle performance, as Lieber and Ward [86] demonstrated comparing the extensor carpi radialis longus and brevis.

1.3.4 Determinants of Power Output

Muscular power, the product of force and velocity, is confined to the maximal force- and velocity-generating capacity of a muscle. Any parameter that can affect V_{\max} and P_o (i.e., the content in the two preceding sections) will have direct implications on the power curve. At either V_{\max} or P_o power is zero, thus peak power occurs at sub-maximal levels of both velocity and force. The velocity at which peak power occurs is defined as optimal velocity (V_{opt}). It has been suggested that optimal velocity may be the most distinguishable feature of the F-V relationship across MHC isoform as there is little, to no overlap of V_{opt} values [111]. Typically there is a ~ 3-4 fold difference in V_{opt} between MHC-I < MHC-II muscle fibers; expressed relatively to V_{\max} , a range of 13% - 28% of V_{\max} [79,111,115,116]. Among the *in vivo* human F-V studies, the relative V_{opt} for whole muscles were consistently ~ 30% of V_{\max} [16-19,99]. The significance of V_{opt} becomes apparent when studies report peak efficiency of muscle fibers occurs at shortening velocities closely approximate to peak power [111,115,116]. Rome [117] explained that the diversity of muscle fiber types allows an animal to move with optimal power and efficiency at any speed (e.g., red and white muscle of fish [118]). The shape of the power

curve is influenced by the a/P_o ratio of the F-V relationship, which is determined by the MHC isoform and temperature. An increase of temperature and/or faster MHC isoforms will result in a higher a/P_o value producing a shallow F-V curve which produces more power [101,119]. Furthermore, the MHC IIA and IIX isoforms have a 5-fold and 9-fold increase in peak power, respectively compared with the MHC I isoform [79,111,115].

Neural contributions to power production in humans include increasing firing frequency, reducing recruitment threshold of high-threshold motor units (i.e., fast twitch muscle fibers), improving muscle coordination, and motor unit synchronization [120]. Overall, the neural contribution to power production is to activate the motor units and muscle fibers more quickly. This will greatly increase the rate of torque development, which is important during ballistic movements (i.e., powerful contractions) [121].

1.4 The Aging Neuromuscular System

Observations of declined neuromuscular function with adult aging have been well documented. In 1989 Dr. Irwin Rosenberg coined the term ‘sarcopenia’; from Greek derivation, sarcopenia translates to poverty (*penia*) of the flesh (*sarx*) [122]. Certainly, the most dramatic effect aging has on the neuromuscular system is a loss of muscle mass, gradual and perhaps functionally minimal until ~60 years of age, but with a more precipitous rate of muscle loss thereafter [123]. Muscle fiber atrophy (type I < type II) and hypoplasia (type I ≤ type II) both contribute to sarcopenia; whether there is a preferential targeting of type II fibers is still inconclusive [60,124-126]. The mechanisms of sarcopenia can be grouped into: hypoplasia-related (immunological, central and

peripheral nervous system alterations and oxidative stress) and atrophy-related (protein metabolism, hormonal, physical inactivity).

With the gradual infiltration of intra-muscular adipose tissue and chronic inflammation associated with aging (increased levels of TNF- α , IL-6, IL-1) apoptotic (programmed cell death) pathways are activated more frequently [127]. Also, oxidative damage from reactive oxygen species can lead to sarcoplasmic and mitochondrial dysfunctions, both having direct implications on apoptosis [128]. Activation of apoptosis will lead to the deterioration of muscle fibers. Alternatively, a reduction of neurotrophic factors and IGF-1 synthesis, leads to neuromuscular junction degeneration [129], which subsequently causes motor neuron decay and abandoned muscle fibers. Neighboring viable motor neurons may acquire the orphaned muscle fibers through collateral re-innervation. This motor unit remodeling has been well documented with electrophysiological studies as well as histochemical studies [130]. However, not all orphaned muscle fibers are re-innervated, leading to hypoplasia. McNeil et al. [131] investigated the number of motor units of the tibialis anterior in young (25y), old (65y), and very old (>80y) men using the motor unit number estimation (MUNE) technique. Their conclusion was motor unit remodelling was able to maintain muscle strength up to 65y, as strength was equivalent between young and old, but the MUNE was lower.

Age-related hormonal and protein metabolism alterations have been associated with sarcopenia which may affect both nerve and muscle tissue or their ability to adapt like younger adults. Physical activity is a major determinant to the quality of life, postponing loss of independence, mobility, and frailty. Many studies investigating the effects of resistance training on muscle mass, strength and power, have demonstrated

improvements in all measurements as well as functional outcomes measurements such as the Short Physical Performance Battery test [16,30,132]. Power et al. [133] established that life-long physical activity could help to maintain the number of motor units compared to healthy controls of the same older age. Mitochondrial function can also benefit from heavy resistance training among older individuals [134]. Although exercise can improve neuromuscular function from the effects of sarcopenia, it cannot eliminate or reverse age-related decrements to the neuromuscular system; but it can provide a greater reserve of functional capacity.

1.4.1 Functional Consequences of Sarcopenia

The most obvious consequence of sarcopenia is decreased muscle strength. However, even though there is a strong correlation between the cross-sectional area of muscle and strength, there appears to be a dissociation of the two during sarcopenia; that is, strength is lost at a greater magnitude than muscular atrophy. From longitudinal studies, it has been estimated that the average annual rate of strength loss after ~ 60 years of age is 2-4% compared to the annual rate of muscle loss of 1% [2,15]. Clark & Manini [135] proposed the term ‘dynapenia’ – poverty of strength – to separate the two different mechanisms occurring during adult aging. This implies a reduced muscle quality associated with aging. Throughout the neuromuscular system there are many sites with which strength can be affected independent of changes in muscle mass: cortical and spinal excitability which will have direct influence on motor unit recruitment and rate coding within the motoneuronal pool [136,137], neuromuscular junction communication [138], excitation-contraction coupling [139], architecture and composition of the

musculo-tendinous complex [140,141], and alterations to the contractile machinery [142-144]. Cross-bridge function and musculo-tendinous architecture both affect force and shortening velocity thus it can be reasonably assumed the age-related alterations to the contractile machinery and architecture will also affect shortening velocity; although these are not always observed to be highly correlated [145].

At the whole muscle, contractile velocity of single twitches have been observed to slow with age, both the rate of tension increase and relaxation [10,27,94,124,146]. The slowing of contractile properties could be attributed to the overall remodelling of type IIX fibers to a slower type IIA or type I, but this is not certain [126]. Architectural data indicate shorter fascicles are associated with muscles of aged individuals [21,63,141]. Shortening velocity is related to the number of sarcomeres in series, and a reduction in these numbers will translate to a slower muscle. However, Thom et al. (2007) normalized V_{\max} to the fascicle lengths in the medial gastrocnemius and eliminated ~50% of the difference between younger and older men, concluding that older had a 16% slower muscle [17]. Thus, impairments to actomyosin interactions likely explain the unaccounted differences. Furthermore, there is much support for intrinsic alterations to the myosin motor being responsible for slower shortening velocities. Indeed, Hook et al. [33] concluded that MHC-I molecules of older humans, rats, and mice were 19-25% slower than their younger counterpart utilizing an *in vitro* motility assay. Possible mechanistic explanations include advanced glycation end products, oxidative stress, and altered protein expression [142,145,147], all of which affect the catalytic domain of the myosin head negatively and subsequently alter the interaction between actin and myosin (i.e., changes the rate constants between cross-bridge states) [142,145,148].

Age-related changes of the F-V-P relationship have been reported for the elbow flexors [18,19], knee extensors [21,22,24,25], plantar flexors [17,20,94] and dorsiflexors [27]; all reporting age-related slower and less powerful muscles. Utilizing Hill's equation, Valour et al. [19] and Toji et al. [18] reported a 31% and 18% reduction in V_{\max} in the elbow flexors, respectively, and Thom et al. [17] reported a 38% slower V_{\max} in the plantar flexors of older compared to younger participants. In addition, peak power was reduced (30%, 45%, and 72%), and V_{opt} was maintained at $\sim 33\%$. The plotted F-V and power curves were consistently shifted down and leftwards with adult aging. The single study which investigated the isotonic [24] and the few that investigated the isokinetic [21,22,25] F-V of the knee extensors did not attempt to model Hill's equation and reported a linear-like relationship. Consequently, there is a need to investigate and characterize the knee extensor F-V-P relationship isotonically and isokinetically, within the same cohort of younger and older participants; not only to fill a void in the literature but also to establish the most appropriate functional basis or modality to explore other aspects of aging such as fatigue.

Many studies of sarcopenia have utilized isometric contractions to investigate neuromuscular properties at rest and during fatigue with an overall agreement of demonstrated fatigue-resistance within the aging muscle [149]. However, there is a growing body of research support strongly linking muscle power with the functional capacity of older individuals, than with isometric strength [14,28,150,151]. Thus, using dynamic contractions to measure power in older participants as well as how fatigue affects muscle power will have important functional implications. Recently, the results of aging and dynamic fatigue on muscle performance have been ambivalent, with reports

supporting less [152], similar [22,64,153], or more [24,45,51,154] fatigue in the aged. The mode of contraction and velocity of the fatiguing contractions may be the primary factors driving these opposing results. Indeed, as muscle efficiency is tightly coupled to the shortening velocity, it is plausible that younger and older muscle attain peak efficiency at different shortening velocities. In consideration of the complexity of the force-velocity-power (F-V-P) relationship, as well as the age-related alterations to the neuromuscular system, there is a need to investigate these properties for muscle groups of key importance for activities of daily living and independence. There are two dynamic tasks commonly implemented to fatigue muscles (isokinetic and isotonic contractions), but there is a paucity of fundamental understanding of how the F-V-P relationship is affected between these two styles of contractions, among aging individuals.

CHAPTER 2

2.0 Introduction

Age-related alterations to the neuromuscular system have been well discussed in many recent reviews [123,124,126,130,135] with the overall conclusion that muscles from older people are weaker, slower, and less powerful when compared to younger adults. Assessing muscle power, rather than maximal strength or shortening velocity alone, provides more insight into muscle function, especially as it is a critical determinant to functional outcomes and autonomy among older individuals [28]. Additional important functional aspects to power are i) intact muscle fibers within a whole muscle operate near conditions of their intrinsic maximal power [117], ii) both power, and the velocity at which maximal power is attained, referred to as optimal velocity (V_{opt}), are highly dependent on the composition of myosin heavy chain isoforms (MHC; I < IIA < IIX) in the muscle [79], and iii) peak mechanical efficiency has been approximated closely to V_{opt} [111,116]. Despite what is known, the majority of age-related investigations into single muscle fiber function have not assessed power. Many studies have reported decreases of 15-34% for specific tension and 20-43% for shortening velocity [1,37,39,41,155], whereas some others have reported a maintenance of muscle fiber function in aged adults [2,40,156].

To assess muscle power the force-velocity (F-V) relationship must be quantified [67]. Among the few single muscle fiber studies to assess age-related changes in power, similar power production for both MHC-I and IIA [40,156] was found, or a 37% decline in muscle power of MHC-II single fibers [155], have been reported. Miller et al. [157] established a correlation between age-related slowing of the MHC-IIA cross-bridge

kinetics and lower whole muscle isokinetic power in humans. However, even with preserved single muscle fiber function, typically the performance of the whole muscle is diminished in healthy [2,40], and mobility-limited older adults [156]. Thus, investigations of *in vivo* F-V relationships in the elbow flexors [18,19], knee extensors [21,22,24,25], plantar flexors [17,20,94], and dorsiflexors [27] all reveal a down and leftward shift of the F-V curve with a range of 20-45% weaker, 12-38% slower, and 38-80% less powerful muscles in the older participants when compared with younger adults. Among the F-V studies cited earlier, both isokinetic and isotonic modalities are used to measure the *in vivo* F-V relationship, however isokinetic movements are artificial in nature compared to isotonic. Many single muscle fiber and whole muscle studies using animals to investigate the F-V relationship do not constrain the muscle isokinetically, but use the isotonic modality in which the load is fixed and velocity can vary during the contraction. Unique to *in vivo* whole muscle, pennated muscles possess a ‘gearing’ property. That is, muscle fascicles may shorten at a different rates compared to the muscle belly and compared to the whole musculo-tendinous unit. Gearing allows the muscle fibers to operate at more optimal lengths and velocities during dynamic contractions (Wakeling 2011 & Azizi 2008). Furthermore, it is known that muscle gearing between isokinetic and isotonic modalities differ which may have functional implications to the whole muscle F-V relationship [87].

Functional studies related to fatigue have found somewhat conflicting results depending on whether the test was isokinetic or isotonic [22, 45]. Thus to better understand the effect of aging on power and to resolve some of the discrepant results found when comparing functional changes such as fatigue, the purpose of this study was to

investigate the effects of age and contraction modality (isokinetic vs isotonic) on the knee extensor force-velocity-power (F-V-P) relationship. To assess the relationship, eight different isotonic loads and eight isokinetic velocities were selected to represent a large range of F-V values within the capabilities of the dynamometer. I hypothesized the older men would be weaker, slower, and less powerful than the younger men, for both modalities, and that the older would achieve maximal power at heavier isotonic loads and slower isokinetic velocities owing to the expected shift to a slower MHC-I type muscle.

CHAPTER 3

3.0 Methodology

3.1 Participants

Eleven younger men (25 ± 4 y) and 11 older men (75 ± 3 y) were recruited from the university community and a local physical activity organization, respectively. All participants were recreationally active approximately three times per week and were not engaging in systematic fitness training; all were free from any neuromuscular or musculoskeletal disorders. The dominant leg was used for testing. Written informed consent was obtained from all participants prior to testing. The experimental protocol was approved by the University of Western Ontario's ethics review board (Appendix C) for experimentation on humans, and the study conformed to the Declaration of Helsinki.

3.2 Experimental Arrangement

A Cybex HUMAC NORM dynamometer (CSMi Medical Solutions, Stoughton, MA; tool kit software, sampling rate at 500Hz) was used to measure torque, angular velocity of the knee joint, and position signals related to the function of the knee extensors during all testing sessions. The dynamometer was adjusted to each subject, aligning the knee to the axis of rotation of the dynamometer. Participants were seated comfortably in the chair and securely fastened with hip, shoulder, and thigh straps to prevent extraneous body movements. The participants were instructed to hold the shoulder straps during all contractions rather than the handle bars of the dynamometer. The hip and knee angles were 100° and 80° , respectively (full knee extension = 180°). The 80° knee angle was chosen to increase the range of motion (ROM) and improve torque recordings to fully engage the lever arm with passive tension with the mass of the shank. The knee adapter

of the dynamometer was secured to the ankle, ~ 2cm proximal the tibial malleolus. The ROM for the dynamic contractions was 70° (80° - 150°), see Figure 3. All knee extensor torques, velocities and positions were sampled at 500 Hz using a 12-bit analog-to-digital converter (Power 1401; Cambridge Electronic Design, Cambridge, UK) and digitized online using Spike2 software (Cambridge Electronic Design). A computer monitor with torque and velocity tracings was provided for visual feedback during all measurements. Additionally, the participants were exhorted during all maximal voluntary contractions.

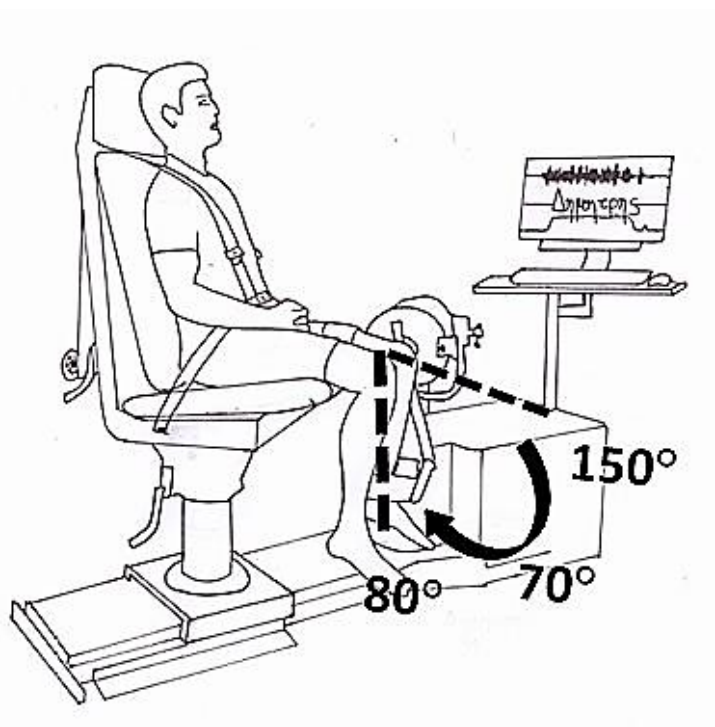


Figure 2. An illustration of the experimental set-up

To assess voluntary activation (VA) and neuromuscular properties of the knee extensors, muscle stimulation was delivered via two custom-made aluminum electrode pads wrapped in a damp paper towel with conductive gel applied liberally. The size of the electrodes varied with respect to each person's thigh size ranging from 6-8 cm in width

and 20-30 cm in length. These electrodes were placed transversally on the thigh; one approximately 6 cm proximal to the patella and the second, 7cm distal to the inguinal fold. Visual inspection and palpation were used to ensure only the knee extensors were activated during electrical stimulation. The electrical stimuli were 200 μ s square wave pulses generated by a computer-controlled stimulator at a maximum voltage of 400 V (Digitimer stimulator, model DS7AH; Digitimer Ltd, Welwyn Garden City, UK). Intensity was controlled by adjusting the current output (mA).

3.3 Experimental Protocol

3.3.1 Isometric Measurements

Electrically evoked and voluntary neuromuscular properties were recorded in the following order: electrically evoked peak twitch (Pt), MCV (2-3x) with superimposed twitch (T_s), resting twitch (T_r), 50 Hz (500ms duration) tetanus. To obtain peak twitches the stimulus current was increased progressively until there was a plateau in torque production. The current was increased an additional 15% to ensure thorough activation of the underlying muscle mass. The MVC was performed at a knee angle of 80° and was maintained for 3-5 seconds with 3 minutes of rest between efforts. Twitches were delivered 2s before, during peak plateau and 2s after each MVC (Figure 2). To assess voluntary activation levels during MVC, the following calculation was performed: $[(1 - (T_s / T_r)) * 100] = \% \text{ activation}$. The stimulation intensity for 50 Hz was increased progressively until an evoked tetanic torque of 40% of MVC was attained. Forty percent of MVC was determined by previous work and pilot testing to be the highest intensity

comfortably tolerated by participants and without activating antagonist muscles using this setup.

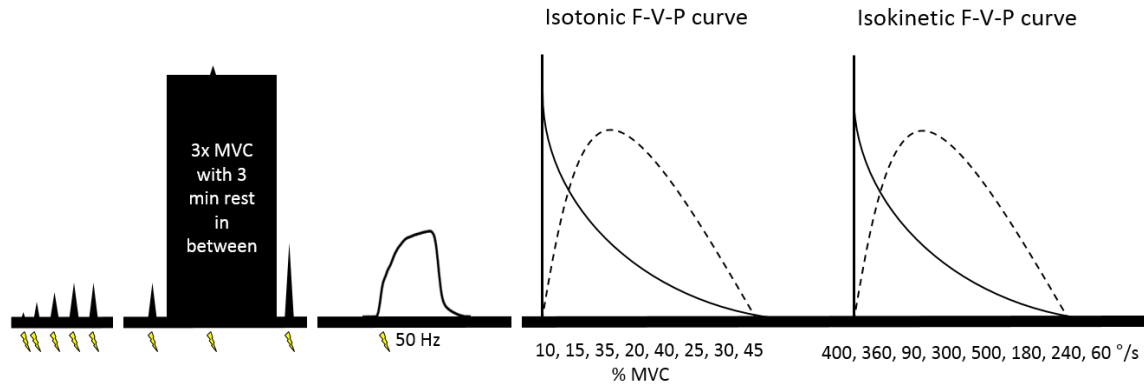


Figure 3. A diagrammatic representation of the experimental protocol

3.3.2 Dynamic Measurements

After 3-5 minutes of rest from the last MVC, 3-5 practice kicks were executed at heavy (~35% MVC) and light (~20% MVC) loads for the isotonic mode and at slow (~90°/s) and fast (~360°/s) velocities in the isokinetic mode. With an additional 5 minutes of rest, participants made 2 rapid isotonic knee extensions at each load (10, 15, 35, 20, 40, 25, 30, and 45% of MVC); and after 5 minutes of rest another 2 rapid isokinetic attempts at each velocity (400, 360, 90, 300, 500, 180, 260, 60°/sec). Approximately 3s elapsed between changes of resistance or velocity (Figure 1). For each participant the order of the resistances (% of MVC) and velocities (°/s) performed were identical (as listed above), providing better performance and reliability during the construction of the F-V curves.

3.4 Data Analysis

The electrically-evoked isometric contractile properties of the knee extensors were assessed by peak twitch torque (P_t), time to peak twitch torque (TPT) – normalized to P_t (TPT/ P_t), as well as 50Hz one-half relaxation time (50Hz-HRT) and 50Hz rate of torque development (50Hz-RTD). Voluntary activation and RTD were measured from the MVC with the highest torque. The chosen RTD was the highest instantaneous rate during the beginning of the 50Hz tetanus and MVC. For the F-V relationships, the effort at each programmed isotonic load and isokinetic velocity with the highest attained instantaneous peak power were selected to extract the torque and velocity, see Figure 4. From these data points a F-V plot was constructed and a curve was estimated using Hill's equation [1] for both isotonic and isokinetic modalities, see appendix A for details, and the predicted maximum shortening velocity (V_{max}) was calculated. The isotonic load and achieved velocity, or the isokinetic velocity and achieved torque that provided the highest maximal power were considered the optimal torque (P_{opt}) and optimal velocity (V_{opt}) values. Furthermore, the P_{opt} and V_{opt} were normalized to their respective maximal parameters (i.e., P_o and V_{max}) to assess the relative shifts of maximal power. The power data were modelled with equation [2] to obtain the power curves, Figure 6.

$$(P+a) \cdot (V+b) = (P_o+a)b \quad [1]$$

$$P = b \cdot P \left(\frac{P_o+a}{P+a} - 1 \right) \quad [2]$$

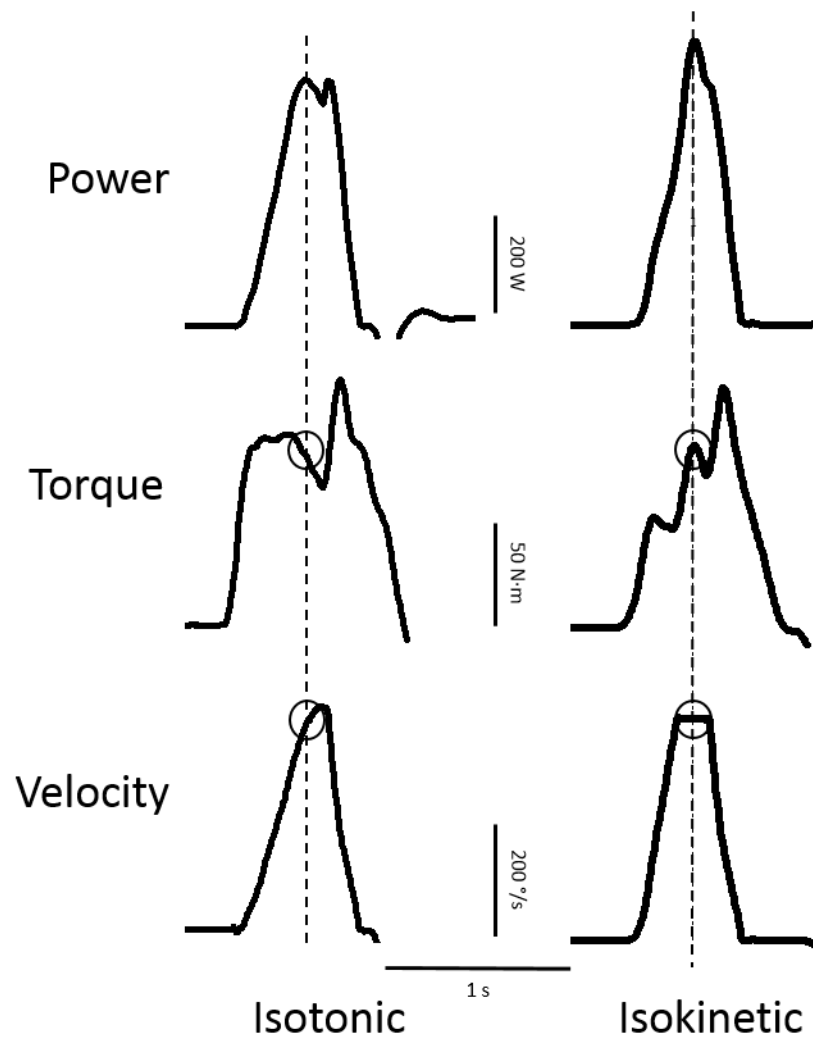


Figure 4. Typical power, torque, and velocity tracings demonstrating the method of extracting the respective values for each modality. Dashed line indicates the values that align with highest instantaneous peak power. The circled areas on the torque and velocity tracings are the values chosen to compare between younger and older men, as well as to construct the F-V curves for each individual.

3.5 Statistical Analysis

Statistical analysis was accomplished using SPSS (IBM, ver. 20). Data normality (Shapiro-Wilk test) and homoscedasticity (Levene's test) were assessed initially. An unpaired t-test was used to compare anthropometric characteristics and contractile data from electrically-evoked twitches (P_t , TPT, TPT/ P_t , 50Hz-RTD, and 50Hz-HRT) and MVCs (peak torque, RTD, and % activation). An unpaired t-test was used to compare younger and older men at each isotonic load and isokinetic velocity separately. A 2x2 (age vs contraction mode) ANOVA was performed to assess maximal power, predicted V_{max} , V_{opt} , and P_{opt} . A Bonferroni post-hoc analysis with an α level of ≤ 0.05 was used to determine statistical significance. All data described in text and tables are mean \pm standard deviation.

CHAPTER 4

4.0 Results

4.1 Isometric Contractile Properties and Strength

The older men (69-81 y) were on average 10 kg heavier than the younger men (19-30 y), with no differences in height (Table 1). Despite similar levels of voluntary activation (VA) (~96%, $p>0.05$), the older men had a 43% weaker isometric maximum voluntary contraction (MVC; Table 2). Compared with the younger men, peak twitch torque, P_t (39%), 50Hz rate of torque development, 50Hz-RTD (30%), and MVC-RTD (53%) were all lower for the older men ($p<0.05$). The isometric contractile speed measures of time to peak tension (TPT) were equal in the younger and older men, however normalized TPT (TPT/P_t), and 50Hz one-half relaxation time (50Hz-HRT) were 57% and 15%, respectively, slower in the older men ($p < 0.05$). The assumptions of data normality and homoscedasticity were met with each comparison.

Table 1. Participant characteristics

	Younger (11)	Older (11)
Age (y)	25 \pm 4	75 \pm 3
Mass (kg)	77 \pm 8	86 \pm 10*
Height (cm)	177 \pm 8	173 \pm 5

Data presented as means \pm SD. * denotes statistical significance

Table 2. Isometric contractile properties of the knee extensors

	Younger	Older	%Δ
MVC (N·m)	272.5 ± 47.4	156.2 ± 24.9*	-43%
VA (%)	96.9 ± 1.8	96.6 ± 3.4	-
MVC-RTD (N·m · s ⁻¹)	1858.1 ± 395.8	874.9 ± 260.0*	-53%
P_t (N·m)	48.3 ± 9.4	29.7 ± 3.35*	-39%
TPT (ms)	96.5 ± 4.2	98.7 ± 3.2	-
TPT/ P_t (ms · N·m ⁻¹)	2.1 ± 0.5	3.3 ± 0.4*	+57%
50Hz-HRT (ms)	117.5 ± 7.10	135.0 ± 6.8*	+15%
50Hz-RTD (N·m · s ⁻¹)	1005.7 ± 256.2	699.5 ± 203.6*	-30%

Means ± SD. %Δ indicates the relative difference for the older compared with the younger men. **MVC** maximal voluntary contraction, **VA** voluntary activation, **RTD** rate of torque development, **P_t** twitch torque, **TPT** time to peak torque, **HRT** one-half relaxation time. * denotes statistical significance.

4.2 Force-Velocity Relationships

During the isotonic contractions, faster knee extension velocities were achieved by the younger men across all loads except at the two heavier loads (i.e., 40 and 45% MVC) compared to the older, see Table 3. Isokinetically, the older men were 40-60% weaker at every velocity ($p < 0.05$) compared to the younger men (Table 4). The older men were not able to attain the $500^\circ/\text{s}$ velocity within the range of motion to produce torque, thus no data were available. The mean predicted isotonic maximum velocity (V_{max}) calculated from each participant using his experimental force-velocity data points fitted with Hill's equation, was 18% slower in the older men. The extrapolated isokinetic V_{max} values were not applicable because the estimations predicted non-physiological values of $1761\text{-}9843^\circ/\text{s}$ for the younger men and $1130\text{ - }6278^\circ/\text{s}$ for the older men.

To assess how the isotonic velocities at each load changed relative to the predicted V_{max} between the younger and older men, the isotonic velocities at each load were compared to their respective isotonic V_{max} . These relative velocities were not different between the two groups. For example, both the younger and older men experienced ~69, 75, and 83% decrease in angular velocity at 35, 40, and 45% MVC, respectively (Table 5A). Conversely, to assess how the isokinetic torques changed relative to P_o (MVC), between the younger and older men, the isokinetic torques at each isokinetic velocity were compared to P_o . The relative torques compared to P_o were similar between the younger and older men for the slower velocities ($60\text{-}240^\circ/\text{s}$), but the older men experienced greater torque decrements at velocities $\geq 360^\circ/\text{s}$, see Table 5B. This comparison provides numerical insight into changes of the shapes of the F-V curves, permitting insight into potential factors responsible for any differences.

To minimize the influence of individual variability on the predictive power of Hill's equation and V_{\max} , the group isotonic means reported from Table 3 and the group isokinetic means from Table 4 were plotted and modelled. The resultant force-velocity curves are found in Figure 3. The predicted group isotonic V_{\max} from the group isotonic force-velocity relationship was $821^{\circ}/s$ for the younger and $606^{\circ}/s$ for the older men, a 26% difference. The predicted group isokinetic V_{\max} from the group isokinetic force-velocity relationship was $1712^{\circ}/s$ for the younger and $1111^{\circ}/s$ for the older men, a 54% difference.

Table 3. Isotonic velocities ($^{\circ}/s$) achieved at each relative load

Isotonic Load	Younger	Older	% Δ
V_{\max}	879 \pm 162 (672-1246)	725 \pm 116 (557-925)*	-18%
10%	448 \pm 33 (402-497)	346 \pm 24 (319-394)*	-23%
15%	415 \pm 33 (362-466)	339 \pm 31 (286-414)*	-18%
20%	394 \pm 33 (335-443)	318 \pm 27 (289-377)*	-19%
25%	346 \pm 31 (304-393)	278 \pm 25 (234-320)*	-20%
30%	296 \pm 33 (252-360)	240 \pm 32 (196-300)*	-19%
35%	260 \pm 34 (213-321)	221 \pm 34 (174-272)*	-15%
40%	207 \pm 36 (148-271)	181 \pm 33 (132-234)	13%
45%	135 \pm 26 (92-171)	128 \pm 33 (79-194)	5%
100%	Zero	Zero	-

Means \pm SD (range of velocities). % Δ indicates the relative difference for the older compared with the younger men. V_{\max} predicted maximal shortening velocity. * denotes statistical significance.

Table 4. Isokinetic torques (N·m) achieved at each programmed velocity

Isokinetic velocities	Younger	Older	% Δ
V_{\max}	N/A	N/A	
500 $^{\circ}/s$	55 \pm 10 (34-74)	---	
400 $^{\circ}/s$	96 \pm 16 (59-121)	38 \pm 14 (24-69)*	-60%
360 $^{\circ}/s$	105 \pm 18 (68-139)	49 \pm 12 (37-77)*	-53%
300 $^{\circ}/s$	116 \pm 23 (74-150)	60 \pm 11 (45-84)*	-48%
240 $^{\circ}/s$	126 \pm 22 (84-164)	67 \pm 9 (55-84)*	-47%
180 $^{\circ}/s$	144 \pm 22 (94-179)	77 \pm 12 (60-104)*	-47%
90 $^{\circ}/s$	192 \pm 30 (120-234)	111 \pm 19 (83-156)*	-42%
60 $^{\circ}/s$	195 \pm 32 (139-251)	117 \pm 27 (83-170)*	-40%
(P ₀) 0 $^{\circ}/s$	273 \pm 47 (173-357)	156 \pm 25 (114-192)*	-43%

Means \pm SD (range of torques). % Δ indicates the relative difference for the older compared with the younger men. N/A not applicable. * denotes statistical significance

Table 5. Relative changes of (A) isotonic velocities to predicted V_{\max} and (B) isokinetic torques to P_o

A Isotonic velocities				B Isokinetic torques			
Isotonic Load	Younger	Older	% Δ	Isokinetic velocities	Younger	Older	% Δ
V_{\max}	100%	100%		P_o	100%	100%	
10%	48 \pm 8	52 \pm 7	4%	500 $^{\circ}/s$	80 \pm 3	N/A	N/A
15%	52 \pm 7	52 \pm 8	-	400 $^{\circ}/s$	65 \pm 3	75 \pm 7*	+10%
20%	54 \pm 8	55 \pm 7	1%	360 $^{\circ}/s$	61 \pm 4	68 \pm 6*	+7%
25%	60 \pm 8	61 \pm 7	1%	300 $^{\circ}/s$	57 \pm 4	62 \pm 5	5%
30%	65 \pm 7	67 \pm 7	2%	240 $^{\circ}/s$	54 \pm 3	57 \pm 5	3%
35%	69 \pm 7	69 \pm 7	-	180 $^{\circ}/s$	47 \pm 3	50 \pm 6	3%
40%	75 \pm 7	75 \pm 6	-	90 $^{\circ}/s$	29 \pm 6	29 \pm 8	-
45%	84 \pm 4	82 \pm 6	2%	60 $^{\circ}/s$	28 \pm 6	25 \pm 10	3%

Means \pm SD. Comparing the isotonic velocities at each load to the isotonic predicted V_{\max} . The younger and older men experience similar relative slowness at each load (A). Comparing the torques at each isokinetic velocity to P_o (maximal voluntary contraction). The older men experience greater relative decrements in torque output at the faster velocities (B). N/A not applicable. * denotes statistical significance.

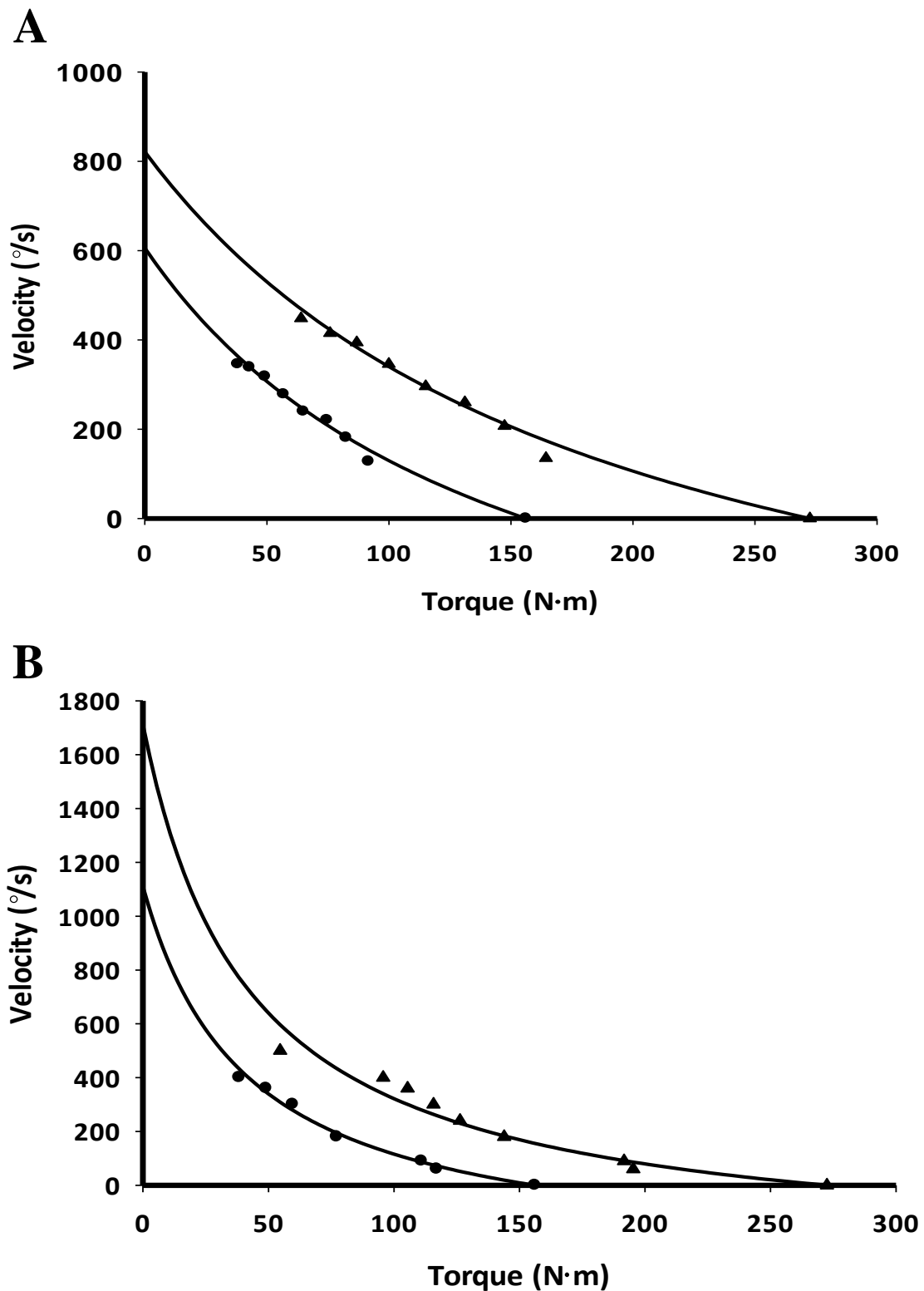


Figure 5. Isotonic (A) and Isokinetic (B) F-V curves for younger (▲) and older (●) men. Shortening velocity was faster for the younger men at light to moderate loads (10-35% MVC) but not for heavier loads (40-45% MVC) (A). The torque produced at each programmed velocity was higher for the younger compared to older men (B)

4.3 Power Curves

The knee extensor maximal power of the older men was ~54% less compared with the younger men in both modalities: isotonic (623 ± 121 W vs 289 ± 51 W, and isokinetically (677 ± 114 W vs 315 ± 57 W). Comparing the power within each age group, there were no differences between the isotonic and isokinetic modalities in both younger (623 W vs 677 W) and older (289 W vs 315 W) men. Across all the isotonic loads the average power was ~50% lower in the older compared to the younger men, and the power profile plateaued between 20-35% MVC for the younger, and between 20-40% MVC for the older men (Table 6A). The isotonic velocities and isokinetic torques reported in Tables 3 and 4 are the associated values with the power data in Table 6. The isotonic torque (optimal torque, P_{opt}) and isotonic velocity (optimal velocity, V_{opt}) achieved at maximum power were 35% (109 ± 22 Nm vs 71 ± 13 Nm) and 36% ($346 \pm 31^\circ/s$ vs $221 \pm 34^\circ/s$, $p < 0.05$) lower compared to the younger men (Figure 5A). In addition to comparing the absolute isotonic torque values of P_{opt} between age groups, when expressed relatively to each individual's P_o , the difference between age was much less, albeit still significant. The majority of younger men attained isotonic peak power at 25% MVC, whereas the older men achieved peak power consistently at 35% MVC, $p < 0.05$. There was a ~10% difference between the younger and older men ($p < 0.05$) in the isotonic V_{opt}/V_{max} ($39.7 \pm 6.2\%$ vs $30.2 \pm 7.5\%$). For the isokinetic power curves, the averaged maximum power across all programmed velocities were 42-58% lower in the older compared to the younger men (Table 6B). The majority of younger and older men attained isokinetic peak power at $400^\circ/s$ and $300^\circ/s$, respectively. Thus, the older reached peak power at a ~25% slower velocity than the younger men, $p < 0.05$. Because V_{max} was

not calculated for each individual's isokinetic force-velocity relationship, a V_{opt}/V_{max} could not be calculated. The P_{opt} was 38% lower in the older men, 96 ± 16 Nm vs 60 ± 11 Nm (Figure 5B), with a similar P_{opt}/P_o of $\sim 37\%$ for both younger and older men.

Table 6. Average maximum power. Isotonic (A) and Isokinetic (B)

A				B			
	Younger	Older	% Δ		Younger	Older	% Δ
10%	501 \pm 77	230 \pm 41*	-54%	500°/s	457 \pm 105	---	
15%	551 \pm 93	253 \pm 39*	-54%	400°/s	677 \pm 114	283 \pm 95*	-58%
20%	599 \pm 109	273 \pm 43*	-54%	360°/s	653 \pm 112	305 \pm 76*	-53%
25%	623 \pm 121	276 \pm 42*	-54%	300°/s	598 \pm 116	315 \pm 57*	-48%
30%	595 \pm 127	270 \pm 45*	-55%	240°/s	523 \pm 91	277 \pm 38*	-47%
35%	596 \pm 129	289 \pm 51*	-52%	180°/s	449 \pm 67	241 \pm 37*	-46%
40%	531 \pm 128	256 \pm 50*	-52%	90°/s	302 \pm 48	175 \pm 31*	-42%
45%	387 \pm 112	200 \pm 53*	-48%	60°/s	206 \pm 34	120 \pm 26*	-42%

Means \pm SD. % Δ indicates the relative difference for the older compared with the younger men. Light grey indicates a plateau in the power curves. Darker grey is the maximum power for each group. * denotes statistical significance

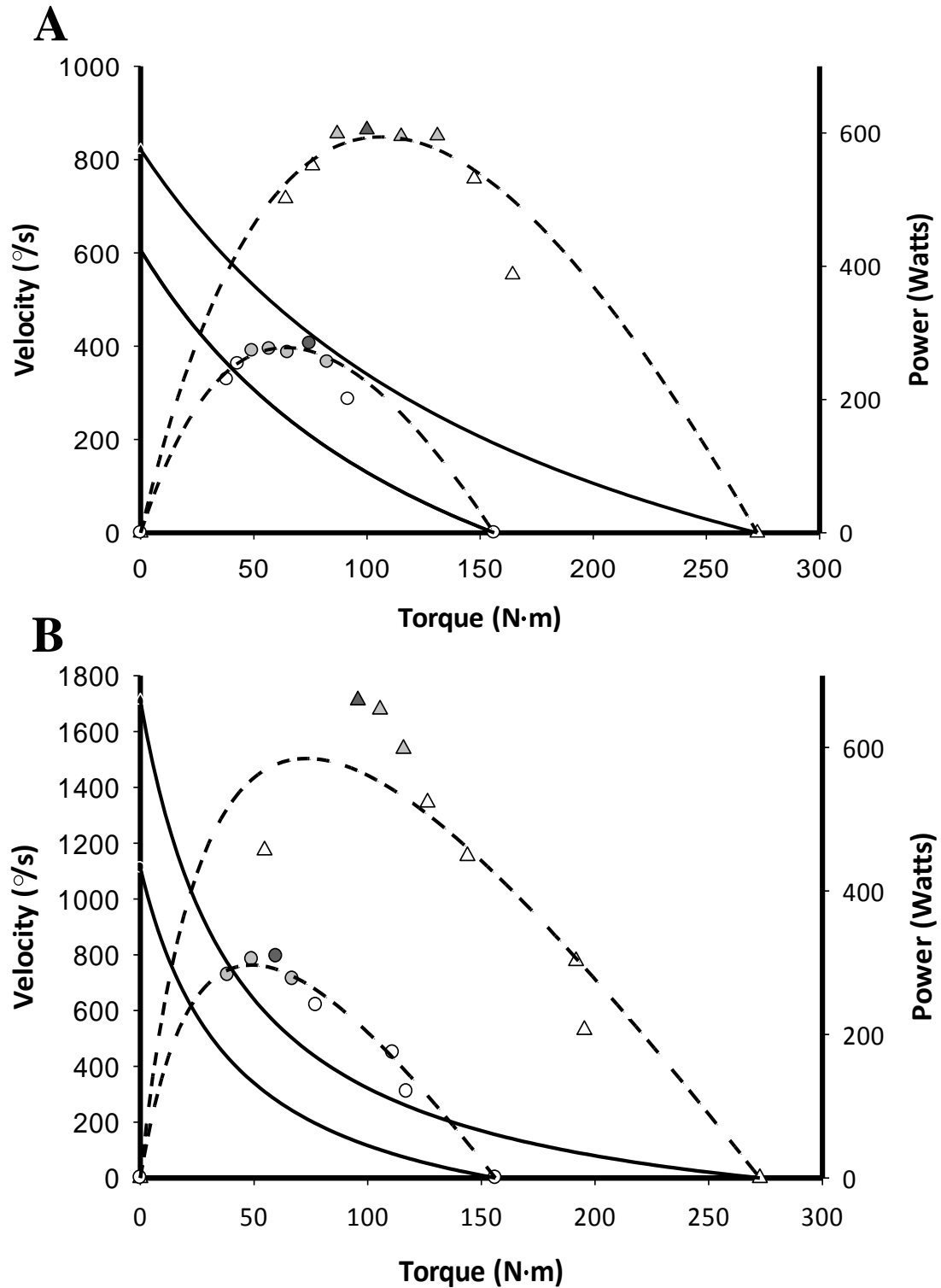


Figure 6. Isotonic (A) and Isokinetic (B) power curves for younger (Δ) and older (○) men. Power was higher in the younger men across all comparisons. Light grey indicates a plateau of power at the crest. Dark grey indicates maximum power. Dashed lines represent the power curves, the solid lines represent the force-velocity curves from Fig 5.

CHAPTER 5

5.0 Discussion

5.1 Purpose and significant results

The main objective was to compare two commonly used modes of dynamic contractions used in assessing muscle performance of the knee extensors of younger and older men. The majority of studies exploring age-related alterations in muscle function have used static (isometric) contractions due to fewer variables to control than those encountered from dynamic actions; such as velocity, work, and power. However, dynamic contractions are an important and understudied component related to muscle function and activities of daily living. The methods used characterized the F-V-P relationship between younger (25y) and older (75y) men and contraction modality (isotonic vs isokinetic). In addition, Hill's equation was modelled to the data to obtain outcome measures describing and quantifying parameters related to force, velocity, and power of the knee extensors. These comparisons provided insight into the value and limitations of each testing modality for describing force, velocity and power.

As expected, the F-V curves of the older men were shifted left and downward, for both contraction types, indicating dynamically weaker (~45%), slower (~20%), and less powerful (~50%) muscles compared to the younger men (Figure 5). The most notable findings relate to the shifts in the F-V and power curves between the isotonic and isokinetic modalities in both younger and older men. For the older men, the optimal torque, P_{opt} , was 35% and 38% lower in the isotonic and isokinetic F-V relationships, respectively, with a modest shift of relative P_{opt}/P_o to heavier isotonic loads compared to the younger men (Table 6A). The optimal shortening velocity, V_{opt} , of the older men was

36% and 25% slower in the isotonic and isokinetic power curves, respectively, with a ~10% lower relative isotonic V_{opt}/V_{max} compared to the younger men (Table 6B). The age-related reduction in isotonic power was affected equally by the weaker and slower knee extensors as shown by the similar reductions in absolute (35% and 36%) and relative (10% and 9%) isotonic optimal torques and velocities, respectively. However, under the isokinetic modality there was a greater reduction in the P_{opt} than V_{opt} for the older men, thus maximum power was affected more by reduced torque. Although the relative isokinetic P_{opt}/P_o was similar between younger and older men, with the difficulties of modelling Hill's equation using the isokinetic F-V relationship, V_{max} could not be obtained and thus relative V_{opt}/V_{max} could not be assessed. Therefore, unlike the isokinetic method, the isotonic modality likely is the better method to test F-V-P relationships in muscle because it more closely represents *in vivo* function and is better suited to modelling using Hill's equation.

5.2 Isometric MVC and Contractile Function

5.2.1 Isometric MVC

The torque recorded from the maximal voluntary contractions, MVC (P_o) of the older men was 43% lower compared to the younger men, corroborating the results from other knee extensor studies (20-48% reduction) [7,9,21,24,25,45,64,158]. The absolute P_o strength of the older men (156 N·m) was on the weaker end of reported knee extensor strength in older men, ranging from 150-210 N·m [16,21,25,45,64,158]. The differences in the age of the older men in this study (75y) and the knee angle tested (80°) compared

to many of these prior studies (67-73y; 90-120°) seems likely to account for these differences. A 12 year longitudinal study of older participants of 71 years of age reported a 2-2.5% annual decrease of muscle strength in the knee extensors [15] and therefore there can be significant differences within a decade of aging for participants over ~65 years of age. It has been suggested the effects of age on muscle architecture could shift the torque-angle relationship (T-A) compared to a younger muscle [140]. However, similar torque-angle profiles between younger and older in the knee extensors have been reported [21,63], with others demonstrating a T-A relationship plateau occurring between 110 – 90° [16,25] and thus our knee angle of 80° is just slightly less than optimal. Utilizing the interpolated twitch technique to assess voluntary activation (VA), the younger and older men were able to activate their knee extensors to a similar degree, ~96%, in agreement with previous reports in the knee extensors [9,45,63] and various other muscles investigated with adult aging [8,10,51,159-161]. Thus, central activation does not appear to be a limiting factor indicating peripheral alterations likely being the predominant source of age-related muscle weakness. These peripheral factors may include reduced overall muscle mass [2,21,38,40,55], selective atrophy of type II muscle fibers [60,162], musculo-tendinous architecture changes [17,140,163], excitation-contraction uncoupling [139], or reduced intrinsic contractile capacity of muscle fibers [42,142].

5.2.2 Contractile Function

As expected, the twitch contractile properties diminished with age and the 39% lower peak twitch torque (P_t) agrees with the results from other studies [9,45,94,161]. Similar time to peak tension (TPT) between the younger and older men also agrees with what

others have reported for the knee extensors [9,45]; although not for all muscles studied [94,161]. However, as TPT is sensitive to the twitch amplitude, normalizing TPT to P_t (TPT/P_t) revealed greater differences of 57% slowing with similar results reported from other studies [45,94]. Also, in agreement with other studies [158,164,165] MVC-RTD was reduced to a greater extent than MVC. Compared with the 30% slower involuntary (stimulated) 50Hz-RTD in the older men versus the younger, the MVC-RTD was 53% lower. Although these findings support generally a large peripheral component to explain age-related changes in muscle contractile function, the greater difference in voluntary (MVC) RTD than that of the stimulated RTD may indicate some central impairment in the older men. Similar average motor unit discharge rates at 100% MVC (~26Hz) between younger and older have been reported for the knee extensors [9], however the initial bursts of impulses could be markedly slower which directly affects RTD [166]. In comparing twitch contractile properties to voluntary muscle performance, one can reasonably associate P_t to the MVC (39% vs 43%), TPT/P_t to shortening velocity (57% vs 21%) and RTD to power (53% vs 53%).

5.3 Force-Velocity Curves

A unique aspect of this study was the construction of the force-velocity-power relationship of the knee extensors using both isotonic and isokinetic modalities in younger and older men. As expected for both groups, there was an inverse relationship with velocity and torque; that is, as the load/torque increased the accompanying velocity decreased [67] (Figure 5). The isokinetically derived F-V curves when modelled using Hill's equation produced a more linear fit whereas most (18/22 subjects) of the

isotonically derived curves yielded the more usual hyperbolic relationship. There is little consistency in the literature regarding the shape of the *in vivo* F-V relationship in human muscles and it is difficult to determine clear reasons for these differences. Despite obtaining varying F-V shapes in the same muscle group between some studies, it is likely that different testing procedures in equipment and methodology, analysis (e.g. angle-specific torque vs. peak torque), and in subject groups (male, female, trained, untrained etc.) may explain this variability among studies. Indeed, angle-specific torque, or mean torque and velocities have been criticized as inappropriate variables to use in the model [100,167], and therefore in this study, the torque and velocity associated with the instantaneous peak power during each knee extension were selected to model the F-V relationships (Figure 4).

Although only two studies, one in younger [44] and one in older [24], used an isotonic test, it seems there is no consensus as to which modality (isokinetic or isotonic) better estimates the knee extensor F-V hyperbolic relationship, and importantly no one previously has compared both modalities in the same subjects, regardless of age. This is important to remove the inter-variability of training status, voluntary activation levels, and whole muscle properties (e.g. muscle fiber composition) encountered when comparing studies. The results in the current study would support the isotonic method as the more appropriate modality as it produced the more usual hyperbolic shape in both younger and older men, and it more closely matches the methods used in reduced preparations upon which the original work is based [67].

5.3.1 Submaximal Loads and Velocities

Indicating a weaker, slower, and less powerful muscle, the F-V curves of the older men were shifted down and to the left compared to the younger F-V curve (Figure 5). Interestingly, for the isotonic F-V relationship, the difference in attained velocity between the younger and older men (15%-23%) remained steady from 10 to 35% MVC load (Table 3), whereas the velocity at heavier loads (i.e., 40% and 45% MVC) were similar between younger and older men. Similar age-load interactions have been reported in other isotonic F-V relationships of various muscle groups such as the plantar flexors [20], and elbow flexors [19,78], while others did not find this interaction for the knee extensors or dorsiflexors [24,27].

Because of the age-related shift of muscle composition to a greater percentage of MHC-I with potential selected atrophy of MHC-II [123], the age-load interaction may be the result of the relatively greater contribution of MHC-I fibers at heavier loads and conversely at lighter loads will reflect the lower amount of MHC-II fibers [44,47]. As there was a consistent ~20% difference between younger and older for isotonic loads among the lighter loads (Table 3), smaller or fewer MHC-II fibers or shorter fascicles (quantitative) [2,40,156] rather than cross-bridge impairment (qualitative) [1,31,33,37,39,42,168] likely explain the results. Furthermore, an age-related impairment of the actomyosin interaction would have manifested through progressively greater relative differences between younger and older with faster velocities, which was not the case as only up to a 4% difference was found across the loads (Table 5A), further supporting a quantitative and not a qualitative impairment.

Conversely, the older men were weaker across all isokinetic velocities with the absolute torque decrements increasing with velocity. These isokinetic torque impairments (40%-60%) were greater than the isotonic velocity impairments, 13%-23% (Table 3 and 4). As a less natural movement isokinetic contractions have limitations that could exacerbate the differences between younger and older men. First, it is difficult to ascertain if the participant is performing a full or maximal voluntary effort as the lever arm will move, whether or not the participant is maximally activated, and secondly, the time to achieve the programmed velocity increases with higher velocities [25,169], and thus the slower contracting older men will achieve velocity further into the ROM than the younger. Although some have reported a similar T-A relationship of the knee extensors between younger and older [21,63], Lanza et al. [25] described an age-knee angle interaction for the knee extensor T-A relationship in that the older men experienced a progressively greater reduction in torque on the ascending limb of the relationship. Thus, the additional time required to reach the programmed isokinetic velocity due to lower firing rates (previously discussed), coupled with being disproportionately weaker at greater knee angles, may largely account for the age-velocity interaction.

No relative differences were found across any of the isotonic loads for the isotonic modality, however, there were relative differences of ~ 7-10% measured for the faster isokinetic velocities (i.e. 360 and 400 °/s), see Table 5. Muscle gearing is the comparison of how fascicles shorten to whole muscle shortening, which allow muscle fibers to function more optimally. Recent investigations into the gearing mechanics of *in vivo* muscles established that older gastrocnemius muscles have slightly less gearing compared to younger muscles [170], and also isotonic contractions maintain better

muscle gearing compared to isokinetic contractions, in particular at faster velocities [87]. It is possible therefore that the older men better maintained muscle gearing isotonically than isokinetically, explaining the age-related relative torque decrements reported in Table 5, in addition to the age-related quantitative changes (e.g., smaller or fewer MHC-II fibers or shorter fascicles).

5.3.2 Maximal Shortening Velocity

Although measured in isometric conditions, the slowing of twitch contractile properties would be expected to manifest itself in the dynamic performance of the whole muscle. The reported mean isotonic V_{\max} for the young men ($879^{\circ}/s$) agrees well with the range reported of the knee extensors isotonic V_{\max} (800 - $950^{\circ}/s$) and isokinetic V_{\max} (600 - $1000^{\circ}/s$) [44,47,99]. No studies have been found estimating knee extensors isotonic V_{\max} in older men. However, our reported isotonic V_{\max} of the older men ($725^{\circ}/s$) is similar to those reporting isokinetic V_{\max} in older participants $\sim 700^{\circ}/s$ [16]. In addition, modelling the group average F-V curved estimated very similar V_{\max} , $\sim 821^{\circ}/s$ for the younger and $\sim 606^{\circ}/s$ for the older. With many of the participants' isokinetic F-V relationship resembling a linear-like shape and poorly modelled with Hill's equation, it is not surprising that the reported V_{\max} values are non-physiological. Even modelling the group average isokinetic F-V curve (Figure 5B) yielded an estimated V_{\max} much faster than what's typically reported.

This is the first study to estimate isotonic V_{\max} , as well as attempt to estimate isokinetic V_{\max} in the same groups of younger and older men. In this study, the isotonic V_{\max} was

18% slower for the older compared with the younger men, in agreement with other reported age-related slowness of V_{\max} , 16-40% for older participants in various muscle isokinetically [17,94] and isototonically [18,19]. Differences among studies may be due to the testing modality, the muscle investigated, and the relative ages and health status. Investigations into single muscle fibers have reported preserved contractile function in both MHC-I & II fibers of aged muscle [2,40], however most report slower intrinsic shortening velocity of fibers ~19 – 50% [1,31,33,37,39,42,168]. Although not systematically strength trained, the older men in this study were a part of a seniors exercise association, exercising 3x/week. Regular exercise can maintain or improve function of single muscle fibers in participants up to 80 years of age [171-173] but perhaps mitigated thereafter [174]. Assuming preserved single muscle fiber quality, an additional explanation for the slower velocities could be changes in the muscle architecture, primarily shorter fascicles. Measured fascicle lengths of the vastus lateralis have been reported to be shorter in older men [21,55,63]. Two studies demonstrated the consequence of fewer sarcomeres in-series by adjusting for fascicle length in the medial gastrocnemius [17] or limb length of the arm [18] eliminating ~50% of the age-related difference in V_{\max} . Also, the age-related muscle composition shifting to greater % of MHC-I will affect V_{\max} as one of the primary determinants of V_{\max} is the myosin ATPase activity [105], which enables MHC-II fibers to be 2-4x faster than MHC-I [175].

The original F-V relationship described by Hill's equation was formulated on isotonic contractions and consequently isokinetic data may not be an appropriate model to explore the F-V relationship and V_{\max} . Indeed, many isokinetic knee extensor studies have experienced difficulties obtaining the classic hyperbolic shape and failed to model Hill's

equation [21,22,25,48,49,90,91,99,100] although some others have been successful in modelling Hill's equation to isokinetic data [16,47,99]. Racz et al. [98] demonstrated that mean torque rather than peak torque as the more suitable input for the isokinetic F-V relationship. As non-physiological estimations of isokinetic V_{\max} were reported in this study from the isokinetic peak torque F-V relationship, it is apparent from Figure 4 of Racz et al. [98] that attempting to model Hill's equation to the isokinetic peak torque F-V relationship would also result in a large overestimation of V_{\max} [98]. An explanation is not readily apparent as to how others have successfully modelled Hill's equation from isokinetic peak torque-velocity data [16,47]. In addition to utilizing peak isokinetic torque to construct the F-V relationship, the discrepancies between the isotonic V_{\max} and isokinetic V_{\max} may be explained by the time-history of muscle length change and level of contractile mechanical state between the two modalities. That is, peak torque occurs earlier for isotonic contractions and later, at more optimal muscle lengths during isokinetic contractions [176].

5.4 Maximum Power, Optimal Velocity and Optimal Torque

Recently, muscular power has been implicated as a better predictor of muscle function that may relate to functional activities in the older populations [14,28,150,151] than either strength or speed. As power is the product of torque and velocity, any of the above mentioned mechanisms that affect MVC (P_o) or V_{\max} will influence age-related differences in muscle power. The results here seem to be the first to compare age-related and modality (isokinetic and isotonic) dependent changes in P_{opt} and V_{opt} . Interestingly,

each group achieved similar maximal power in both isotonic and isokinetic modalities (Younger - 623 vs 677W; Older - 289 vs 315W), respectively.

Furthermore, both isotonic and isokinetic power curves displayed the parabolic shape with left and downward shifting in the older men. As muscle power is largely determined by the muscle fiber type, with a possible 9-fold difference among the MHC isoforms, $MHC-IIx > MHC-IIa > MHC-I$ [111], it was expected that the younger men would be more powerful compared to the older men. The absolute knee extension maximal power matched those reported using iso-inertial [24] and isokinetic contractions [40], and the relative decrease of ~54% agrees with the range of relative power decrease of 31-72% reported for various muscles [17,19,20,25,78,94,177,178]. The relative changes of power across all the isotonic loads were consistently ~ 50% lower in the older men, however there was an age-velocity interaction for the isokinetic power curve (Table 6). That is, there were progressively greater decrements in power at faster isokinetic velocities. It is unclear why this occurs for isokinetic and not isotonic contractions, but it is likely related to the less natural isokinetic movement and the way in which age and muscle gearing (discussed previously) interact during each modality. With similar reductions of ~36% in both isotonic P_{opt} and V_{opt} for the older men, it appears impairments in torque and velocity equally affect the isotonic maximum power. This is further supported by equivalent 10% differences between younger and older men for relative isotonic V_{opt}/V_{max} (40% vs 30% V_{max}) and P_{opt}/P_o (25% vs 35% MVC), respectively. This equivalent contribution of torque and velocity resulting in lower power in the older men may be due to the quantitative changes to the muscle architecture, i.e., smaller fibers, as well as shorter fibers. These relative shifts to a slower velocity and higher torque agree

with others who investigated the effects of aging on the elbow flexors and plantar flexors [19,20,95] using an isotonic modality. During a leg press movement, Yamauchi et al. [23] and Allison et al. [166] concluded that the slower velocities contributed significantly greater to the lower power than torque. However, both studies did not normalize the V_{opt} to V_{max} , which provides a better basis for comparisons and thus more insight as to how power is influenced by relative changes of intrinsic shortening velocity capacity.

The older men here experienced greater reductions in isokinetic P_{opt} (38%) compared to V_{opt} (25%) which extends what was found during isokinetic testing in the triceps surae [17,20]. More importantly, the V_{opt} achieved here during isokinetic testing was 16% and 36% higher for both younger and older men, respectively, compared to the isotonic V_{opt} . An explanation is not immediately obvious as to why the isokinetic modality have higher V_{opt} compared to isotonic V_{opt} , however the differences in muscle gearing may explain this. During isotonic contractions, muscle fibers generate torque to exceed the opposing load, as this is occurring, the series elastic component (SEC) is being stretch and the fibers are shortening; which may favor a V_{opt} at a slower velocity. In contrast, isokinetic contractions generally do not stretch the SEC at the initiation of movement, which may favor a faster V_{opt} . Once again it is clear that the two testing modalities are not comparable and the system and the age-related changes respond differently to each method. In addition to the altered gearing dynamics between isokinetic and isotonic contractions [87], the nature of an isotonic contraction engages the whole musculo-tendinous system before movement occurs, thus the contractile and elastic components contribute to the generation of power. Although the isokinetic contractions do not take advantage of the elastic properties of the system for optimal power production they still

achieve slightly higher power, ~8%. Thus, the time-history of muscle length change and level of contractile mechanical state experienced during isokinetic contractions may offset the reduced muscle gearing. This would be expected to maintain similar power production to isotonic contractions at faster velocities.

5.5 General Conclusion

In summary, sarcopenia significantly affects the function of the knee extensors with ~50% less power due to a combination of weaker (43%) and slower (20%) muscle compared to a younger muscle. It appears from the main results in Tables 3, 4, and 5 that the older men in this study primarily experienced quantitative impairments (e.g., smaller or fewer MHC-II fibers or shorter fascicles) with no alteration to cross-bridge function. It is clear from the results that the response of the age-altered system is not the same at each testing modality and this may explain some of the conflicting reports in the literature concerning F-V-P relationships. Although there are some merits in utilizing an isokinetic modality perhaps especially for rehabilitation, the isotonic modality should be the preferred modality to explore physiologic changes using more natural dynamic movements features which also relate more closely to modelling using Hill's equation to estimate V_{\max} . In addition, the isotonic modality allows the experimenter to assess changes in velocity throughout various interventions, which is often regarded as the critical determinant of power. These shifts in optimal torque and velocity, not only across age but also between modalities, may begin to clarify the equivocal reports of aging and muscle performance.

5.6 Limitations

Although the knee extensors were 43% weaker in the older men compared to the younger, architectural measurements of the knee extensors would have provided more direct and additional insight into muscle quality. The measurements of muscle physiological cross-sectional area, fascicle length, and muscle volume would allow more accurate comparisons of torque, shortening velocity, and power, respectively. Additionally, muscle biopsies, although more invasive, would have provided valuable information to the potential differences in muscle composition (myosin heavy chain isoforms) commonly reported between younger and older participants. The normalization of muscle function to the architecture and quantification of the whole muscle composition together would allow a more complete understanding of the influence of all aspects related to muscle function observed in the older participants.

Fitting the human to the dynamometer always results in some testing limitation related to joint angles, range of motion and the ability of the dynamometer to adjust and cope fully with fast angular velocities. The ROM was 70° (80-150°), which proved to be limited for faster knee extensions of >400°/s isokinetically and <15% MVC isotonic load. Thus the isotonic loads and isokinetic velocities selected were limited to the recording capacity of the Cybex HUMAC NORM dynamometer. The maximum measurable velocity of 500°/s was limiting for the younger men but was not a factor for any of the older men. Measuring additional knee extensions faster than 500°/s would provide better modelling power for Hill's equation. Also, utilizing isokinetic peak torque rather than mean torque

did not allow a direct measure of V_{\max} , although peak torque was the appropriate measure to compare with isotonic contractions as both are related to instantaneous peak power.

During an isometric MVC, the results showed that both younger and older men could equally activate their knee extensors maximally, to ~96%. However, of particular importance is the ability to assess activation during a dynamic contraction. I did not have any measure of activation during dynamic contractions. Some have implemented the ITT during isokinetic contraction assessing any changes in the torque tracing [179,180]. On the other hand, ITT is difficult to apply during isotonic contractions as the torque is constant, thus perhaps velocity or acceleration must be monitored. Using surface EMG to assess voluntary activation might have been helpful to relate to neural drive, but this is problematic during dynamic contractions [181]. Finally, current results are applicable only to the sample population studied and so for example women and frail elderly might not respond in the same manner as healthy younger and older men.

5.7 Future Directions

The relationship between peak power and mechanical efficiency provides an opportunity to investigate *in vivo* muscle fatigue. Measuring the torque (P_{opt}) or velocity (V_{opt}) at peak power provides an approximation of the contractile function associated with peak efficiency for the muscle. Accounting for the *in vivo* shifts of whole muscle peak power allows appropriate contraction parameters specified for each participant during a fatiguing protocol. Having each participant performing repeated maximal knee extensions at their peak power parameters will eliminate unaccounted variability with

different participants working at different efficiencies. Many studies have demonstrated that sarcopenia affects men and women differently. Thus to be complete in our efforts to comprehensively assess age-related changes to the F-V-P relationship of the knee extensors, the inclusion of women is necessary. Lastly, an investigation of the effects of training at maximal power parameters, with continual monitoring of any potential shifts in the power curve during training, would be particularly useful, to assess how it affects the F-V-P relationship. Any training regime that provides superior improvements to muscle performance among seniors has great implications to their quality of life.

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APPENDICES

Appendix A. Mathematical modelling of Hill's equation

$$(P+a) \cdot (V+b) = (P_o+a) b \quad (1)$$

Estimating the maximum velocity at zero load (V_{\max}) involved transforming equation (1) into a linear equation to calculate the 'a' and 'b' constants. A demonstration of the linearization of (1):

Expand the equation

$$(P + a)V + Pb + ab = P_o b + ab \quad (2)$$

cancel common variables

$$(P + a)V + Pb = P_o b \quad (3)$$

rearrange

$$(P + a)V = (P_o - P)b \quad (4)$$

express P as a function

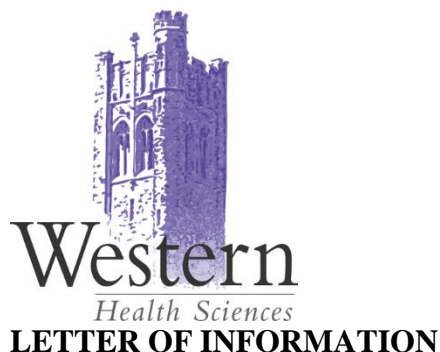
$$P = \frac{P_o - P}{V} b - a \quad (5)$$

Plotting P v.s $\frac{P_o - P}{V}$ enables a least-square linear regression to be fitted to the data, producing the slope (constant 'b') and y-intercept (constant 'a'). Using SigmaPlot (version 12.0, Systat Software, Inc, Chicago, IL) the data were fitted with Hill's equation to predict V_{\max} . Muscle power (force (N·m) · velocity (rads/s)) from

$$\text{Power} = bP \left\{ \frac{P_o + a}{P + a} - 1 \right\} \quad (6)$$

was modelled to create a power curve for each F-V curves.

Appendix B Letter of Information



Neuromuscular control of human movement.

Study Investigators

Dr. Charles Rice	Professor	Ph.D
Dr. Tim Doherty	Physician	Ph.D, MD
Cameron Smith	Graduate Student	B.Sc
Brad Harwood	Graduate Student	MHK
Geoffrey Power	Graduate Student	M.Sc
Matti Allen	Graduate Student	M.Sc

Introduction

You are invited to participate in a research study examining the effects of prior muscle contraction on neuromuscular activity.

Summary of Research

The objective of this study is to quantify the relationship between prior muscle contraction and neuromuscular activity during static and dynamic movements in young and older adults.

We are interested in assessing changes in the nervous control and muscle responses of your limb muscles following different types of muscular effort. Prior to and following a high intensity, short duration (5-10 seconds) muscle contraction, followed by a fatiguing (longer duration) muscular contraction we will measure both voluntary and involuntary muscular contractile properties during static (no joint movement) and dynamic (joint movement) movements.

Data from this study will help in understanding how age and activity influence human motor control and function.

You may participate in this study if you are a:

- Healthy male or female (18-100 years).

You CANNOT participate in this study if you:

- Have cardiovascular or respiratory disease that limits daily activities.
- Cannot tolerate the exercise protocol (experience undue discomfort).
- Have any neuromuscular limitations.
- Are, or may be pregnant.
- Are osteoporotic.
- Are taking medication(s) known to influence motor cortex excitability or muscle function (e.g. benzodiazepines, neuroleptics, anticonvulsants, muscle relaxant, calcium channel blockers).

Description of Research

If you are eligible and agree to participate in this study, you may be requested to come to the Neuromuscular Laboratory at the Canadian Center for Activity and Aging in the Arthur & Sonia Labatt Health Sciences Building (room 311) for up to seven separate visits over the course of a six month period. Each visit will last up to 3 hours. During the testing portion of each visit, you will be secured in a seated or lying position. Up to seven different muscle groups may be tested, of the following:

1. Dorsiflexors (muscles that lift the toes and flex the ankle) on the front of the leg
2. Plantar flexors (muscles that point the toes and extend the ankle) on the back of the leg
3. Elbow extensors (muscles that straighten the elbow) on the back of the arm
4. Elbow flexors (muscles that bend the elbow) on the front of the arm
5. Knee extensors (muscles that straighten the knee) on the front of the thigh
6. Knee flexors (muscles that bend the knee) on the back of the thigh
7. First dorsal interosseous (muscle that pulls the index finger towards the thumb) on the hand

For all measures either part of your arm or leg will be secured and supported in a simple device that will measure the strength of your muscle contractions. Testing of the dorsiflexors and plantar flexors will occur with your ankle secured to a support that will measure the force and speed of contraction. Assessment of the elbow flexors and extensors will be performed in the seated or lying position with your arm supported under the elbow and the wrist secured to a strength measuring device which will measure muscle contraction. The knee flexors and extensors will be assessed in a seated position with your leg secured to a lever arm for measurement. The first dorsal interosseous will

be tested in a seated position with your arm and wrist secured to a platform and your index finger secured to a lever arm.

During your visit we will test your maximal strength during voluntary (under your control) and electrically stimulated (involuntary) muscle contractions. During voluntary efforts, electrical activity (electromyography) of the nerves that control your muscle will be measured using small needles or wires inserted through the skin (intramuscular) into the muscle belly and with surface electrodes stuck to your skin over the muscle. For involuntary measures the nerve to your muscle will be electrically stimulated with a brief (less than 2 second) pulse from pads taped on the skin over the muscle. The stimulation feels like a mild to moderate tingling sensation.

Surface electromyography will be recorded from electrodes (adhesive pads) on the surface of your skin over the muscle belly and is not associated with any discomfort. Intramuscular electromyography requires brief insertion into the muscle of a small diameter hypodermic needle containing a very small (100 μm , approximately the diameter of a human hair) wire electrode. The penetration of the skin feels like a pin prick and requires no anesthetic or special preparation aside from a thorough cleansing of the skin with alcohol. Following insertion, the needle may be removed and one end of the wire recording electrode remains inside the muscle until completion of the test. Alternatively, depending on the task (i.e. high intensity fatigue protocol), a slightly larger diameter ($\sim 125 \mu\text{m}$) recording needle may be left in the muscle for several minutes while you perform a series of muscle contractions.

Muscle fatigue will be induced by having you perform moderate to strong contractions in either a continuous or intermittent pattern. Fatigue tasks will last between approximately 30 seconds and 30 minutes. Muscle strength will also be measured by having you perform a brief maximal contraction of up to approximately 10 seconds duration. Strength and contractile properties (as described above) of the muscle will be assessed at regular intervals during and following the fatigue and potentiation tasks.

During select testing sessions, an ultrasound unit will be used to take an image of your muscle through the skin to determine muscle size and shape. This procedure uses sound waves projected through the skin and reflecting off of muscular structures; it is non-invasive, is not associated with any pain or discomfort and is not known to carry any risk to the subject.

Risks and Discomforts

You may experience some mild muscle soreness for 1-2 days following each testing session.

Electrical stimulation is achieved with isolated and grounded electrical stimulators designed specifically for humans. The sensation of electrical stimulation of your nerve or muscle, though not painful, may be uncomfortable for some participants due to the unfamiliarity with such a procedure.

A very low risk of infection (less than 1:10,000) is associated with intramuscular electromyography. The risk is greatly reduced by standard sterilization procedures and proper clinical practices including alcohol cleansing and the use of latex gloves. In addition to standard sterilization procedures and proper clinical practices, new needles will be used for each subject to better eliminate the risk for infection. Mild soreness and redness may be present at the insertion site following the testing session, but it is generally short in duration. If soreness or redness persists for an extended period of time at the insertion site following the testing session, the subject should contact the study investigators or a physician immediately.

For standard diagnostic ultrasound, there are no known harmful effects for human subjects.

Benefits

It is unlikely that you will benefit from participating in this study, however the information collected may help better understand nerve and muscle changes associated with contractile history.

Voluntary Participation

Participation in this study is voluntary. You may participate, refuse to answer questions, or withdraw from the study at any time with no effect on your academic standing or employment status. The investigator has the right to withdraw you from the study at any time.

Confidentiality

If you agree on the consent form, full names and phone numbers will be collected and retained for contact regarding future studies and/or follow up from representatives of the Canadian Centre for Activity and Aging research laboratories located in the Arthur and Sonia Labatt Health Sciences Building. All study results are coded to protect your identity and privacy. All hard-copy data are placed in a locked cabinet and are only accessible to study investigators. Electronic records will be encrypted, password protected, and stored on a computer with appropriate security firewalls. Only grouped data (averages and ranges, etc.) will be used in any subsequent presentations or publications. Representatives of the University of Western Ontario Health Sciences Research Ethics Board may contact you or require access to your study related records to monitor the conduct of the research.

Compensation

You will be compensated for travel and parking costs for each visit, as appropriate (up to a maximum of \$25.00) per testing session.

Summary

You should keep this letter of information for future reference. If after reading this you agree to participate, you will be asked to sign a consent form saying that you have read this letter of information, and that your questions have been answered. If you choose to sign the consent form, you will also receive a copy of your consent to retain in your personal records. You do not waive any legal rights by signing the consent form.

What to do if you have questions or problems

If at any stage during the testing you have questions or problems you should address these to the study investigator.



Project Title: Neuromuscular control of human movement.

Informed consent:

I have read the accompanying letter of information, have had the nature of the study explained to me, and I agree to participate. All questions have been answered to my satisfaction.

_____	_____	_____
Print Name	Signature	Date

I agree to allow my data to be used in other studies and to be contacted regarding future investigations.

_____	_____	_____
Print Name	Signature	Date

Signature of Person Obtaining Consent

_____	_____	_____
Print Name	Signature	Date

Appendix C Ethical approval documentation



Use of Human Participants - Ethics Approval Notice

Principal Investigator: Dr. Charles Rice
Review Number: 18097
Review Level: Full Board
Approved Local Adult Participants: 100
Approved Local Minor Participants: 0
Protocol Title: Neuromuscular control of human movement
Department & Institution: Anatomy & Cell Biology, University of Western Ontario
Sponsor: Natural Sciences and Engineering Research Council

Ethics Approval Date: July 22, 2011

Expiry Date: August 31, 2015

Documents Reviewed & Approved & Documents Received for Information:

Document Name	Comments	Version Date
UWO Protocol		
Letter of Information & Consent		

This is to notify you that the University of Western Ontario Health Sciences Research Ethics Board (HSREB) which is organized and operates according to the Tri-Council Policy Statement: Ethical Conduct of Research Involving Humans and the Health Canada/ICH Good Clinical Practice Practices: Consolidated Guidelines; and the applicable laws and regulations of Ontario has reviewed and granted approval to the above referenced study on the approval date noted above. The membership of this HSREB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

The ethics approval for this study shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the UWO Updated Approval Request form.

Member of the HSREB that are named as investigators in research studies, or declare a conflict of interest, do not participate in discussions related to, nor vote on, such studies when they are presented to the HSREB.

The Chair of the HSREB is Dr. Joseph Gilbert. The UWO HSREB is registered with the U.S. Department of Health & Human Services under the IRB registration number IRB 00000940.

Signature

Ethics Officer to Contact for Further Information

<input checked="" type="checkbox"/> Janice Sutherland	<input type="checkbox"/> Grace Kelly	<input type="checkbox"/> Shantel Walcott
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This is an official document. Please retain the original in your files.

CURRICULUM VITAE

Name: Justin Roland Paturel

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2011-2014 M.Sc. Kinesiology

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The University of Western Ontario
2011-2013

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2009-2011

Publications:

Paturel, J.R., Power, G.A., Makrakos, D.P., Vandervoort, A.A., and Rice, C.L. 2013 Age-related effects of isotonic and isokinetic fatigue when matched for peak power in knee extensors. *Med. Sci. Sports and Ex.* (Suppl. 45, No. 5, S126)

Paturel, J.R., Power, G.A., and Rice, C.L. 2012 Repetitive isotonic contractions are more fatigable compared with isokinetic when matched for peak power. *Appl. Physiol. Nutr. and Metab.* 37: Supplement 1, S27-S28.

Choi, I., Allen, M.D., Smith, C.B., **Paturel, J.R.**, and Rice, C.L. 2012 Neuromuscular fatigue of the knee extensors in coronary artery disease patients: a pilot study. *Appl. Physiol. Nutr. and Metab.* 37: Supplement 1, S6-S7.

Power, G.A., **Paturel, J.R.**, Rice, C.L., and Vandervoort, A.A. 2012 Increased residual force enhancement following damage. *Proceedings of the 17th Biannual Conference of the Canadian Society for Biomechanics*, Burnaby, BC. June 6-9. Poster session 2, #066.